

AN INVESTIGATION INTO THE INCREASED MORTALITY
FROM PULMONARY TUBERCULOSIS SINCE 1914.

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The writer has for the past nine years devoted his time to the study of chest affections, mainly tuberculosis, and has had varied experience, as Resident Medical Officer at the Royal Victoria Hospital, Edinburgh, in the Paddington and Kensington Dispensary for the Prevention of Tuberculosis, London, in the Birmingham Municipal Anti-Tuberculosis Centre, and, for the past seven years, as Medical Superintendent of Romsley Hill Sanatorium, Birmingham, with 140 beds for the treatment of Pulmonary Tuberculosis.

It seems astounding that mental and venereal diseases appear to excite such an abnormal amount of attention from the general public, the legislators, publicists, and physicians, whereas the more dangerous tuberculosis, with its high mortality and train of ill-health and hardship, is only now receiving a small share of the notice it undoubtedly deserves. Perhaps its very antiquity accounts for this attitude of "laissez-faire." We are told that in the year 400 B.C. Hippocrates drew a clear picture of the disease; so that probably, in those days also, tuberculosis was a

well-known and widespread disease, and as time went on came to be considered an evil which had to be borne.

The country generally is overwhelmed by an epidemic like the recent influenza scourge; and yet tuberculosis has for centuries taken its toll of human and animal life unchecked, and has only evoked a "Kismet" from the great majority of our fellows. But when the death-rate was found to be diminishing, interest was awakened, and prophecies of a comparatively speedy and entire disappearance of the disease from amongst us were general.

Powell and Hartley ("Diseases of the Lung", page 386) say in regard to this diminishing death-rate:-
 "It is comforting to find statistical evidence that, with improved sanitation and a general increase in the wage-earning capacity of the labouring classes, there has been a marked and continuous decline in the death-rate from this disease. This is well brought out in the seventy-first Annual Report of the Registrar-General, whereby it will be seen that the death-rate from Phthisis, and also from all forms of Tuberculosis, has been steadily decreasing. Fifty years ago, the annual death-rate from consumption was 26 per 10,000 living, between two and three out of every 1,000 people dying annually of the

disease. In 1908 it was only 11.15, barely more than one person in every 1,000 falling a victim."

Unfortunately, since 1914, the curve of the death-rate from Pulmonary Tuberculosis has shown an alarming upward inclination, so that I have felt it my duty to investigate, and if possible determine from clinical evidence, the probable cause of this rise, and the deduction of future prognosis.

Let us first of all consider, that, for fifty years prior to 1914, there was an almost continuous decline in the death-rate from Pulmonary Tuberculosis in the British Islands, and, at the end of fifty years, the death-rate was reduced by 50%, as shown in the subjoined tables.

Crude Annual Death-Rate per million living from
Pulmonary Tuberculosis in England and Wales.

<u>Five years</u>	1871-75	=	2218
	1876-80	=	2039
	1881-85	=	1830
	1886-90	=	1635
	1891-95	=	1462
	1896-1900	=	1322
	1901-1905	=	1215
	1906	=	1157
	1907	=	1148
	1908	=	1123
	1909	=	1091
	1910	=	1015
	1911	=	1061
	1912	=	1021
	1913	=	989
	1914	=	1022
	1915	=	1136
	1916	=	1156 (civilians only)
	1917	=	1228

Taking the figures from the County Borough of Smethwick of 80,000 inhabitants, whose cases of Pulmonary Tuberculosis are treated in the Sanatorium of which I have charge, the same alarming increase is seen.

Death-rate from Pulmonary Tuberculosis per 1,000 living (calculated per 1,000,000).

<u>Year</u>	<u>Death-rate per 1,000 living.</u>	<u>D.R. per 1,000,000</u>
1897	0.83	830
1898	0.66	660
1899	0.81	810
1900	0.74	740
1901	0.87	870
1902	0.75	750
1903	0.65	650
1904	0.93	930
1905	0.73	730
1906	1.03	1030
1907	0.78	780
1908	0.86	860
1909	0.82	820
1910	0.84	840
1911	0.94	940
1912	0.90	900
1913	0.87	870

<u>Year</u>	<u>Death-rate per 1,000 living.</u>	<u>D.R. per 1,000,000</u>
1914	1.15	1150
1915	1.10	1100
1916	1.20 (Civilians only)	1200 (Civilians only)
1917	1.30 " "	1300 " "
1918	1.40 " "	1400 " "

There is small wonder, then, that the optimistic views held a few years ago are not so pronounced today. In fact, in some quarters optimism has given place to pessimism, as may be judged from the daily press, e.g., in the following paragraph from "The Birmingham Daily Post" recently:-

"Increasing Tuberculosis.

At a meeting of the Worcestershire Insurance Committee on Saturday, Dr. Dixey, who deals with the Tuberculosis reports, said that "unfortunately the disease was increasing. There were 60,000 discharged soldiers who were suffering from it, and he was getting very pessimistic about it all."

Early Diagnosis and Possible Pitfalls.

It would be helpful to review the clinical pictures presented by this disease, Pulmonary Tuberculosis.

In "The Lancet", December 13th., 1919, there is an annotation "Wanted a Pathognomonic Sign of Pulmonary Tuberculosis." Conflicting statements by eminent physicians are reviewed, and comment is made on the unsatisfactory state of affairs in the diagnosis of Pulmonary Tuberculosis. Such an article, in a leading medical journal, serves to bring home to us the difficulties in diagnosing the disease. Personally, I consider that, in a malady of such varied aspects, it is too much to expect a sign of absolute infallibility. It has been said that every case of Pulmonary Tuberculosis is a law unto itself, and the greater the experience one has with the disease, the more true does this statement become, and it is only by wide experience, and a properly balanced judgment of the varied pictures presented, that accuracy in diagnosis and prognosis can be obtained.

The following examples are interesting from this standpoint.

(a) In several cases where I was unable to find any physical signs in the chest, I found tubercle bacilli in the sputum.

(b) I examined a girl and found crepitations over both lungs, persisting for twelve months. She developed acute appendicitis, and died after the operation. At the post-mortem examination, a very few mediastinal glands were the only signs of a tubercular infection.

(c) I recently saw a man, diagnosed as a case of Pulmonary Tuberculosis, with a history of wasting, and with well-marked crepitations at both pulmonary bases, who on examination was found to have carcinoma of the pylorus.

(d) Another man, sent into the Sanatorium with what appeared to be well-marked physical signs of pulmonary tuberculosis, was found to have most perfect bronchial casts in his sputum - a case of fibrinous bronchitis.

(e) Another case - a woman - came under my notice, with very much enlarged glands in the neck, and a history of wasting. The temperature was swinging in character through two or three degrees daily. After a long time, and after treatment by various methods - tuberculin included - had been tried in vain, the glands suddenly became painful and disappeared within three days. The temperature was still variable and intermittent, and the patient's greatest complaint was weakness. On making a thorough examination, there was a slight degree of jaundice noticed, the

spleen was enlarged and appeared about twice its normal size, the blood-count being normal. In consultation with Dr. J. G. Emmanuel, the case was diagnosed as Familial Acholuric Jaundice.

(f) Recently I admitted to the Sanatorium a man with a history of haemoptysis, who was diagnosed as a case of pleurisy, with effusion, probably of a tubercular nature. A sample of the pleural effusion was found to be bloodstained, and made me suspicious of the cause of the effusion. Cytological examination of the pleural effusion and X-Ray examination of the chest failed to give any material help in the differential diagnosis between a tubercular condition and a malignant condition. The liver was found slightly enlarged, and rapidly became more enlarged, and in three weeks from date of first examination filled practically half of the abdominal cavity. Post-mortem: - sarcoma of the lung was found.

These few examples show that Pulmonary Tuberculosis resembles Hysteria. A complete examination of the whole body must be made, before arriving at an accurate diagnosis. It has been said that the most important point in diagnosis is to know when to suspect Pulmonary Tuberculosis. Symptoms and physical signs

of other pulmonary affections may closely resemble those produced by Tuberculosis, as in the case of Sarcoma already quoted, and the numerous cases of so-called chronic Bronchitis and Asthma, which are often found to be tubercular in origin. Truly, the way of the clinician who aims at accurate diagnosis, is beset with difficulties.

Clinical Pictures.

Modes of Entry of Tubercle Bacilli into Lung and Type of Infection.

Let us review the modes of entry of the tubercle bacilli into the lung and subsequent changes produced.

Heller's Classification is as follows:-

- | | |
|--|---|
| <u>A. Primary "inhalation"</u>
Pulmonary Tuberculosis | { <ul style="list-style-type: none"> (1) of the alveoli (2) of the bronchioles (3) of the lymphatic follicles of the lung |
| <u>B. Secondary Tuberculosis</u>
of the Lung. | { <ul style="list-style-type: none"> (4) by inhalation (5) through the blood-stream (6) by the rupture of a lymphatic gland into the air passages (7) <u>through the lymphatics</u> |

No.7 appears to be the most common method of infection, as has been maintained by Sir Robert Philip. A pharyngitis of varying degree is a most common sign in cases of pulmonary tuberculosis - in fact, it is the exception to see a really healthy-looking pharynx among consumptives.

The infected glands in the triangles of the neck, small and shotty to palpation, are an almost constant accompaniment to apical tuberculosis, and in all probability mark the paths of infection. I have often observed, in cases where these cervical glands have become markedly enlarged and gone on to caseation and abscess formation, that the apex on the same side is not affected to anything like the same extent as ~~that~~ on the opposite side of the body .

When the tubercle bacillus has entered the lung, the most common sites of the growth of the tubercles are in the bronchial wall -

(1) Sub-epithelial,

(2) Peri-bronchial

(Bandelier & Roepke, "A Clinical System of Tuberculosis", page 19)

This early deposit of tubercular tissue is of importance when we come to discuss the early physical signs of pulmonary tuberculosis.

In reviewing the clinical pictures presented by Pulmonary Tuberculosis, two sets of observations must be made:-

- (1) The amount of pathological change in the lungs.
- (2) The amount of general toxaemia.

It is surprising how far the second set - the most important both from diagnostic and from prognostic aspects - have been ignored. A very exhaustive work, "The Early Diagnosis of Tubercle", by Rivière, has recently appeared, and it is remarkable that, in such a book, practically no mention is made of the toxaemia of tuberculosis.

The disturbance of the patient's general health, to my mind, forms a very large factor in diagnosis, and the principal factor in determining treatment and prognosis. It is the measure of the patient's response to the toxaemia. The patient's general health is, after all, the determining factor in estimating working capacity, and loss of the feeling of well-being is a common cause of people seeking medical advice. The ill-health, and consequent inability to work, caused by pulmonary tuberculosis probably affects at least seven times the number of persons who die of the disease.

Perhaps the discovery of the tubercle bacillus by Koch has drawn more attention to the local lesions caused by tuberculosis, and has overshadowed the more apparent general signs of the malady.

A most important point in prognosis is the previous duration of symptoms of general toxaemia, as it indicates the resisting power of the patient. Thus, a man with a history of ill-health for say a year, if found to be, at the time of examination, in a similar state of health to another man, who gives a history of three months' duration, has a much better prognosis than the latter.

A fuller explanation of the general toxaemia of tuberculosis will be made later.

Symptoms.

The first point in the diagnosis is the history given by the patient.

As one might expect, these symptoms fall into two classes:-

- (1) Local,
- (2) Constitutional.

The local symptoms are:-

- 1. Cough.
- 2. Sputum.
- 3. Haemoptysis.
- 4. Pain.
- 5. Dyspnoea.

None of these symptoms is pathognomonic of pulmonary tuberculosis.

The cough is usually worse morning and evening, and the same remark applies to the patient's expectoration. A history of coughing was given in 98% of the cases admitted to the Sanatorium. Cough, of course, is not a symptom confined to pulmonary tuberculosis, but in that disease it is usually of a softer character than the cough met with in bronchial affections. The cough tends to become harder in character as the sputum diminishes.

Sputum.

All cases of pulmonary tuberculosis do not exhibit this symptom. In a series of 590 cases admitted to Romsley Hill Sanatorium, sputum was present in 81.35% of the cases which were of stages II and III of Turban Gerhardt classification, and the following is the result as regards presence or absence of tubercular bacilli.

	<u>Tubercular Bacilli present</u>	<u>Tubercular Bacilli absent</u>	<u>No Sputum</u>
<u>Males</u>	184	177	43
<u>Females</u>	32	87	67
	<u>216</u>	<u>264</u>	<u>110</u>

The method employed in staining for the bacillus was the Ziehl-Neelsen process, used on smears made with a stout platinum loop. Concentration methods were also employed

in doubtful cases.

The simplest, and one of the most useful concentration methods I have used, is the carbolic acid method. The sputum is mixed ^{thoroughly} with twice its own quantity of 1 in 20 carbolic acid solution. A pipette with a rubber teat is used to suck up as much as possible, and the pipette and teat are fixed in a test-tube stand over a test-tube. At the end of 24 hours, the heavier deposit has sunk to the nozzle of the pipette, and the first two or three drops are used to make the smear for staining. I have found this method quite as satisfactory as the more complicated Ellerman and Erlandsen. Personally, I prefer to examine directly, by the Ziehl-Neelsen method, repeated specimens, in preference to the concentration methods.

It is admitted that cases where tubercle bacilli are present in the sputum are open cases of pulmonary tuberculosis, but the absence of the bacillus in the sputum does not preclude the possibility of the presence of the disease. In one case I had under my care, a girl with signs of vomica formation, I made repeated examination of the sputum, with the anti-formin method, without discovering any tubercle bacilli - until one day I found quantities.

When we remember that in a chronic abscess, such as is present in pulmonary tuberculosis, the bacilli are in the abscess walls, and not in the centre of the pus as in an acute abscess, we can explain the failure to find the bacillus in many cases even where marked physical signs are present in the chest. It can be demonstrated that the tubercle bacilli are in the abscess walls, because pus in an abscess of tubercular origin is often found to be sterile. The abscess does not heal when the pus is evacuated in many cases, unless either the walls are scraped (when the bacilli may be found), or some antiseptic, like iodoform, is used to disinfect the abscess cavity.

Morphological Differences in Tubercle Bacilli.

In the human tubercle bacilli, differences in form and retention of Ziehl-Neelsen stain are met with. As a rule, the smaller, deeply-staining kind are found in more active cases than the longer, faintly-staining variety.

The evidence of phagocytosis of the bacilli by leucocytes is always a favourable sign. I have not observed phagocytosis to be a common phenomenon in sputa examined.

Number of Tubercle Bacilli present.

I made a set of observations to determine, if possible, a relationship between the number of bacilli present in the sputum and the type of case in regard to response to treatment.

Scale.

T.B.+ = Average of 1 or less Tubercle Bacillus per
microscopic field.

T.B.+ + = Average of 1 to 3 T.B. per microscopic field.

T.B.+ + + = Average of over 3 T.B. per microscopic field.

From my observations, cases under the heading T.B.+ + seemed to respond better to treatment than cases T.B.+ , and certainly much better than T.B.+ + + .

Mixed Infection.

The presence of other organisms, associated with the tubercle bacillus, has an effect on the prognosis of a case of pulmonary tuberculosis. Whether the importance attached to this by R. W. Allen is merited, is a matter for controversy. The streptococcus certainly, as a companion to the tubercle bacillus, is a serious complication. The staphylococcus, pneumococcus, and micrococcus catarrhalis are fairly commonly met with, and do not appear to me to have any markedly detrimental effect on the progress of the case.

Haemoptysis.

Haemoptysis is a symptom which is not pathognomonic of pulmonary tuberculosis, and is met with in cases of cardiac disease, aneurisms, and malignant lung conditions. In the majority of cases, however, the cause is pulmonary tuberculosis. The amount varies greatly, and may be only a few stained sputa, or a haemoptysis of pints. A history of haemoptysis was given by 48% of the patients admitted to the Sanatorium. There is undoubtedly a seasonal incidence of haemoptysis, and the majority of copious haemoptyses I have seen have occurred in the months of October to April. This is probably accounted for by catarrhal processes assisting tubercular ulceration.

Pain.

A history of thoracic pain was given in 76 per cent of the cases examined. It is often surprising to find well-marked pleural crepitus present in cases of pulmonary tuberculosis, where the patient affirms that there has never been any pain experienced. In cases where pain does occur, it is usually of a relapsing nature, and is apt to recur at intervals without any apparent extra exciting cause.

Dyspnoea.

This symptom is not altogether due to local causes, but, I consider, is also due to the systemic intoxication. A history of dyspnoea was given in 92 per cent of the cases examined.

The dyspnoea may be only experienced after exertion. It may assume an asthmatic form, and be specially marked at night. Sudden attacks of dyspnoea always give rise to a suspicion of progressive disease. I consider also that toxic action on the heart is a large factor in producing shortness of breath in pulmonary tuberculosis, and in many cases produces distension of the heart, with functional bruits.

Mention must be made of mechanical causes of dyspnoea, as in pleurisy with effusion, which occurs in about 1 per cent of Sanatorium cases.

Constitutional Symptoms.

Apart from dyspnoea, which, as already indicated, is partly constitutional, three symptoms undoubtedly of a toxic nature are commonly met with:-

- (1) Weakness,
- (2) Loss of weight,
- (3) Night sweats.

Weakness and loss of weight are present in practically all cases admitted to the Sanatorium, varying of

course very much in degree, and by no means in a constant ratio to the amount of physical signs present in the chest. Night sweats were present in 70% of the cases admitted. Night sweating is probably the symptom which is above all others peculiar to pulmonary tuberculosis.

Other symptoms, such as vomiting, may be present. Vomiting is often mechanical, and a resultant of stimulation of the pneumogastric nerve by cough.

From the symptoms alone it would be quite inaccurate to diagnose pulmonary tuberculosis, but they are invaluable in awakening suspicion of the disease. Sometimes symptoms may be so slight as to be unnoticed by the patient. I saw a striking illustration of this when I was assistant Medical Officer at the Paddington and Kensington Dispensary. I examined a girl aged twelve years, who showed signs of early pulmonary tuberculosis. As is the custom, an examination of all the members of the household was made. The father said that he had never had a day's illness and did not wish to be examined. After some persuasion, he consented, and was found to have active pulmonary tuberculosis, with quantities of tubercle bacilli in his sputum, of which he had never taken any notice. Again, it was quite a common

occurrence in the early days of the war to get patients sent to the Sanatorium who were discovered to have pulmonary tuberculosis at the medical examination for the Army, and who had never suspected that they had any chest affection.

Examination of the Patient.

Having had suspicions aroused as to the probable existence of pulmonary tuberculosis, it is now the Clinician's duty to examine the patient.

A general examination should first be made, and note made of the presence of pallor, cyanosis, signs of dyspnoea and clubbing of the fingers. This will be more fully dealt with later.

An examination of the pharynx should be made. As already stated, it is rare to find an absolutely healthy pharynx in cases of pulmonary tuberculosis.

Examination of the Chest.

Inspection.

The position of the patient for this examination is most important. He should be stripped to the waist, and the nether garments should be fastened as loosely as possible round the waist. The patient should stand in an easy position, with muscles relaxed, and with the arms hanging by the sides and the head erect. It is quite useless to attempt to make an accurate examination for signs of early pulmonary tuberculosis with

the patient in bed - he should stand in a good light.

Three sets of observations should be made:-

1. Form of chest.
2. Movement of chest.
3. Abnormal appearances of a trophic nature.

I always inspect the patient's back in the first instance for signs of any kyphosis or scoliosis, and for marks of any previous operations or wounds, which may affect the contour or movement of the chest. Slight loss of contour, or flattening at an apex with deficient movement, I have come to regard as a very certain sign that there is disease in the underlying lung. In more advanced disease, especially in healing cases, the flattening may be so marked that the sternal end of the clavicle on the side is prominent - in fact, there is slight dislocation of the sterno-clavicular joint.

Abnormal appearances of a trophic nature will be discussed later under Integumentary System. Palpation I have used to confirm inspection.

Percussion.

Percussion is of two types -

Gentle,

Strong.

I use the middle finger of the left hand as the

pleximeter, and the middle finger of the right hand as plexor. Direct percussion is used over the clavicles, the skin being fixed by the index and middle fingers of the left hand. Percussion is from the wrist. In the first instance, gentle percussion is used, under one ounce in force.

As one of the early manifestations of pulmonary tuberculosis is a peri-bronchitis, which damps the resonance of the lung tissue, gentle percussion must be used, so that the resonance of the adjacent healthy lung tissue may not overshadow impairment of resonance over the affected area. If strong percussion is used, the presence of disease may be thus masked.

Signs of Impaired Resonance.

In percussion of the front of the chest, the patient stands as for inspection, while in percussion of the back, he stands with his arms crossed over his chest, each hand on the opposite shoulder, and his head slightly bowed. Percussion note is a relative quantity, and thus impairment of the note is likewise relative.

In impaired percussion note, two qualities are present:-

1. A higher pitched note than over other parts of the lung.
2. The feeling of resistance in the percussing finger.

I have recognised three degrees of impaired resonance.

1. Percussion Note - 1 = impairment of percussion note

definite to gentle percussioin, but indefinite when stronger percussioin used.

2. Percussion note - 2 = impairment definite both to gentle and slightly stronger percussioin.

3. Percussion Note - 3 = impairment marked and also resistance to plessor finger. Note obtained such as that over liver and as in cases of pleurisy with effusion.

Topographical Percussion.

Mention must be made of Krönig's isthmus of resonance, which is well described by Rivière in his "Early Diagnosis of Tubercle", page 66 et seq. This is a refinement of Philip's method of percussing the apices measured in finger-breadths above the clavicle. Tidal percussioin is used to determine the expansion of the bases, and was first described by R. W. Philip. Percussion of the lowest limit of the lung is made at the end of expiration, and the limit marked by a skin pencil. The patient then takes a deep inspiration, and again the lowest limit of lung resonance is noted. In healthy adults there is a difference of $1\frac{1}{2}$ to 2 inches between the two marks.

Rivière calls attention to bands of impaired resonance, which he says occur regularly in pulmonary tuberculosis, in the supra-scapular spaces and in the mid-

interscapular spaces ("Early Diagnosis of Tubercle", page 39). I have been unable to find these bands in a very large proportion of my cases of undoubted pulmonary tuberculosis. In the cases where I have found these bands of dulness, X Rays have shown some alteration in the hilus of the lung. Rivière's bands were present in 20% of the cases examined in the Sanatorium.

Auscultation.

Type of Stethoscope.

Personally I use a phonendoscope, and apply the chest piece firmly, but without any marked pressure, to the patient's skin. I think the type of stethoscope is immaterial, provided the clinician becomes accustomed to and uses one stethoscope.

Results of Auscultation.

Here I have followed Professor Wyllie's scheme of recording breath sounds and accompaniments, as detailed in Hutchison and Rainy's "Clinical Methods."

Granular Breath Sounds.

Rivière calls attention to breath sounds in early tuberculosis which he describes as granular. These are probably produced by breaking-down of the peri-bronchitic nodules. I have often observed the presence of fine crepitations round the heart apex beat, which I regard as catarrhal in origin.

Generalized Bronchitic Rhonchi may mask breath sounds and other accompaniments. In such cases, variations in the percussion note become most important, as in a simple bronchitis no alteration of percussion note occurs. Localized Rhonchi are suspicious signs of a tubercular lesion, and I have frequently observed Rhonchi replacing crepitations in cases where healing is taking place in the lungs.

Classification of Cases on Pathological Signs.

The basis of most classification schemes is the time-honoured Turban-Gerhardt scale, with its stages I, II, III.

Stage I.

Disease of slight severity, limited to small areas on either side, which in the case of infection of both apices does not extend below the spine of the scapula or the clavicle - or, in the case of affection of the apex of one lung, does not extend below the second rib in front.

Stage II.

Disease of slightly more marked severity, more extensive than Stage I, but affecting at most the whole of one lobe - or slight disease, extending to the second interspace on both sides and without any serious tuberculous complications.

Stage III.

All cases of greater severity than Stage II, all those with considerable cavities, and all cases with grave tuberculous complications.

In recording pathological signs, I have used the graphic method of Sir R. W. Philip.

It is rare to find a case where only one apex is affected, and, in the great majority of instances, one apex is more seriously affected than the other. An analysis of 200 consecutive cases admitted to Romsley Hill sanatorium showed:-

Apex more seriously affected

<u>Right</u>	<u>Left</u>	<u>Base</u>
38%	61%	1%

In the cases where the base was seriously affected, histories of pneumonia as a commencing factor were generally given. My experience of basal cases is that they do not respond well to treatment. The probable reason why the left apex is more often affected than the right, is that the left lung has not the same free expansion as the right, on account of the mediastinum and heart being deflected to the left. This induces a certain amount of stasis, which favours the growth of the tubercle bacillus.

The Systemic Intoxication of Pulmonary Tuberculosis.

When one speaks of the Systemic Intoxication of Pulmonary Tuberculosis, an explanation is required. Tuberculosis of other parts of the body - bones, glands or skin - does not give rise to the same constitutional disturbance as pulmonary tuberculosis. This might be ascribed to the close association with highly vascular structure in the pulmonary type. I have seen cases where there was extensive bone and gland mischief, and yet no appreciable systemic disturbance was present. Rivière and Morrand describe such cases as "localized tuberculosis".

Now, in every case of pulmonary tuberculosis, there exists a certain amount of general toxaemia, varying in intensity in different individuals, marked in some cases, slight in others, but always present to a less or greater degree. The toxaemia of pulmonary tuberculosis is not a general systemic intoxication, but an intoxication which follows certain very definite lines, and which is selective in its action. As far as I can judge, the following systems are attacked:-

- (1) The Circulatory System,
- (2) The Integumentary System,
- (3) The Neuro-Muscular System.

(4) The Glands.

(5) The Mental Balance.

The Circulatory System.

As regards the blood, no gross change is apparent. From 500 blood examinations I could find no marked change, either in the haemoglobin or in the number of red blood corpuscles, to suggest an anaemia either of the "chlorotic" or of secondary type. The majority of cases of pulmonary tuberculosis present a certain amount of pallor. This pallor is not due to blood changes, and must be ascribed to other causes, probably changes in the integumentary system.

Changes do occur in the blood; for example, Arneth has sought to establish a method of diagnosing tuberculosis by a differential leucocyte count.

Complement Fixation and Opsonic Index.

As diagnostic signs, these are both unreliable in Tuberculosis, as, according to Osler, 90% of the inhabitants of the British Islands are affected by the disease. I do not intend entering into discussion on these points, but simply mention these phenomena as indications that pulmonary tuberculosis causes changes in the blood.

Blood Pressure.

From my own observations, I am convinced that a definite lowering in the blood pressure occurs in pulmonary tuberculosis. This is probably due to toxic action on the heart and blood vessels, and I believe that a considerable number of cases of so-called D.A.H. are simply tachycardia cases, secondary to tubercular toxaemia.

The Integumentary System.

Undoubtedly pulmonary tuberculosis gives rise to changes in the skin. In a case of moderate disturbance, a skilled observer will note that the skin of the consumptive differs from normal in various ways. There is a loss of lustre in the consumptive skin, or, if one might so describe it, a certain wilting of the skin. The glow of health is absent, and in some cases, the skin, particularly over the chest, has an appearance of white marble in its intense whiteness. This change is all the more striking in a man of the labouring classes, where one would expect to find a brawny chest. In other cases, the skin appears greasy and unhealthy looking - often it will be finer in texture than normal, and in many cases large blue veins are seen shining through the integument. Another change which seems to be fairly constant in the skin of

the consumptive, is the presence of little varicose plexuses, which are seen mostly in the cervical and dorsal regions. These little spider nervi seem to indicate some interference with the blood-supply of the skin.

The Skin Appendages.

The Hair.

The hair shows alterations in pulmonary tuberculosis. The consumptive has usually a good head of hair, especially the consumptive woman. Abundant growth of hair in the axillary region is also a feature in pulmonary tuberculosis. Growth of hair on parts of the chest where hair is not usually found in healthy people, may be noted in consumptives. I have noticed that often, over the site of the pulmonary lesion, there is either a little tuft of hair or several scattered hairs. This is specially noticeable in cases with basal lesions. The explanation of this abnormal growth of hair I consider to be that it is due to stimulation of the sebaceous glands and hair follicles by the tubercular toxin.

Night Sweats.

Night sweats in an adult are almost symptomatic of pulmonary tuberculosis, and are probably due to some

abnormal stimulus to the sweat glands. One is often impressed, on examining a consumptive, by finding active perspiration in the axilla - beads of sweat are seen running down the arms and chest wall.

The Neuro-Muscular System.

Sir Robert Philip has pointed out that tuberculosis is essentially a neuro-muscular poison. This can be seen by the undue response of the muscles to slight stimuli, and is commonly known as myotatic irritability. It is elicited either by firmly tapping the skin with the finger, over the pectoral region especially, or, better, by gently drawing the finger over these areas, when contraction of the underlying muscle fibres takes place. When this is more marked, a distinct swelling of the muscle fibres may be seen, following the finger tract (myoedema) .

While myotatic irritability cannot be said to be pathognomonic of pulmonary tuberculosis, yet it is an excellent indication of ill-health, and forms a measure of the degree of toxaemia present.

The myotatic irritability is not always equal on the two sides of the chest, and is usually more marked on the more affected side, showing that a certain

amount of local toxaemia occurs.

There is also present an undue dilatation of the cutaneous blood-vessels, resulting in some cases in dermographia. This indicates a loss of nerve control on the vessel walls. The muscles to the touch have a feeling of soapiness, due to loss of muscle tone. The loss of weight which accompanies the disease is not only due to a loss of subcutaneous fat, but there is also muscular wasting - atrophy and fatty degeneration (Bandelier and Roepke). This, to my mind, is one of the strongest arguments in favour of the action of the tubercular toxin on the muscular system.

Glandular System.

Enlargement of lymphatic glands is due to toxic action, and is seen in the triangles of the neck in practically all cases of apical tuberculosis.

Thyroid Gland.

Turban draws attention to a slight swelling of the thyroid gland, as one of the earliest symptoms of tuberculosis, more seldom seen in later cases. This enlargement of the thyroid is especially seen in young women, and may reach such a grade that, in conjunction with the tachycardia frequently present in tuberculosis, it may lead to

an erroneous diagnosis of exophthalmic goitre in cases of early or latent phthisis. We must, however, not forget that many French authors, and some German, consider tuberculosis to be a cause of Graves's Disease (Bandelier & Roepke, "A Clinical System of Tuberculosis", page 46). Comparison of the signs of exophthalmic goitre with those of the systemic intoxication of pulmonary tuberculosis reveals many points of similarity, but I have never seen a case of pulmonary tuberculosis with the eye phenomena of Graves's Disease, although I have noted many consumptives with varying degrees of enlargement of the thyroid gland.

Neurosis and Psychoneurosis in Tubercular Persons.

This is a very wide subject, on which much work has still to be done. The "Spes Phthisica" is a well-known phenomenon, but it is only one of the many signs that tubercular toxin affects the mental balance. Neurasthenia and Tuberculosis are often very closely allied, and irritability of temper is a very common feature of the consumptive, especially the cases of early disease.

The effect of Mental Disease on Tuberculosis.

It is a well-known fact that mentally afflicted

people are specially liable to develop pulmonary tuberculosis, which is the terminal condition in a large proportion of asylum patients.

Temperature and Pulse.

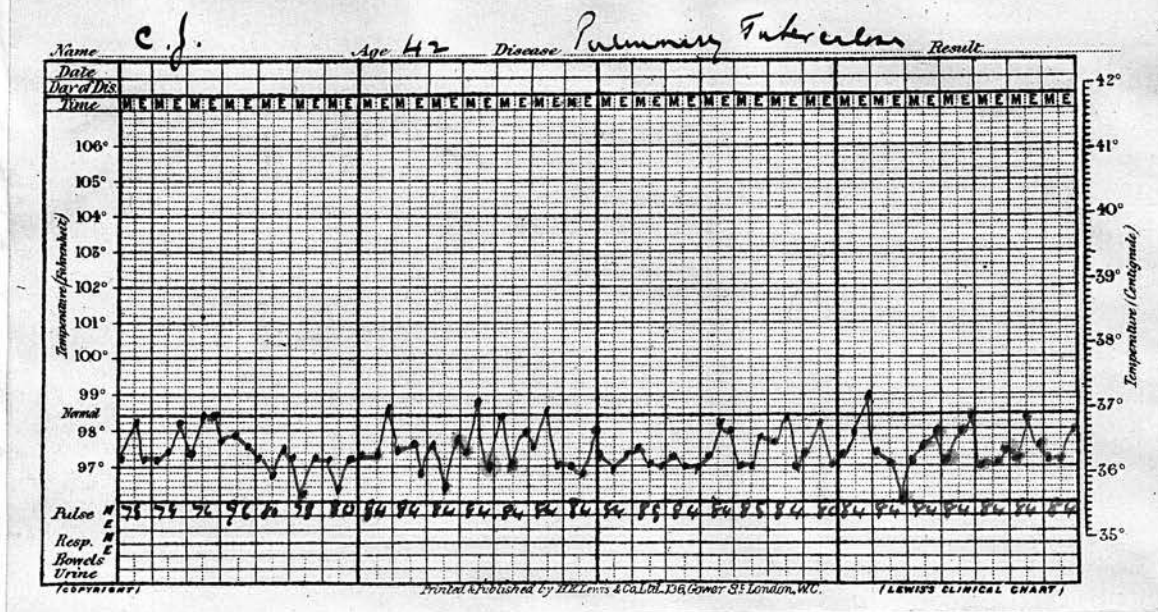
The two great general indications of toxæmia have yet to be discussed, viz., the temperature and the pulse rate. The pulse is perhaps more important than the temperature in pulmonary tuberculosis. Alterations in the pulse rate, especially tachycardia, often occur with a temperature not above the normal line, and in such cases, if there is any marked degree of tachycardia, the case is usually one of a progressive character, in which the temperature centres are incapable of action, owing to toxic paresis of the heat-regulating mechanism. Tuberculosis without tachycardia, or at least instability of the pulse, is exceedingly rare (Fishberg).

Many of the so-called cases of D.A.H. I consider to be due to tubercular toxæmia. This instability of the pulse rate is a specially marked feature of the cases of tuberculosis met with in discharged soldiers, the pulse rate being often as high as 140 per minute, without any marked temperature change, and indicates a toxæmia to which the resistance is inadequate.

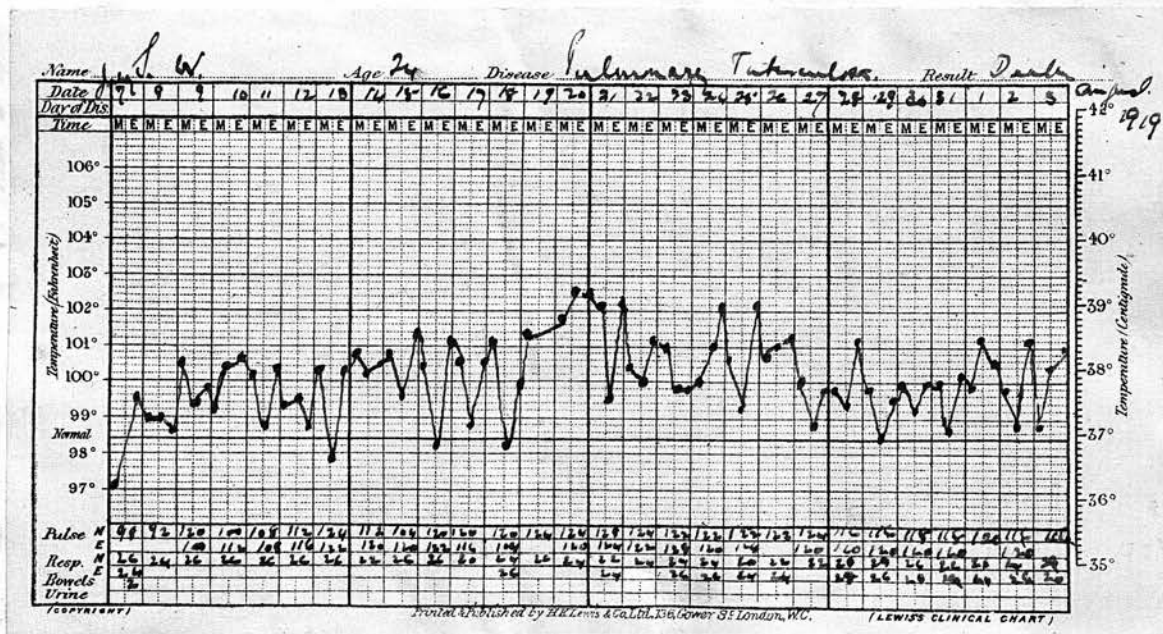
The Temperature.

The temperature in pulmonary tuberculosis does not conform to a constant type. Undoubtedly, extreme sensitiveness of the body temperature is characteristic of tuberculosis, but except for the frequency of a raised evening temperature as compared with that of the morning, and that not always constant, no characteristic feature of the temperature seems to exist. Cases may run a long course and end fatally, without any marked rise of temperature, especially where there is little power of resistance. I have seen many such cases in the past twelve months, and in each one the pulse rate was accelerated, running about 116 per minute. The respirations have been varied, from 16 to 30 per minute, thus demonstrating that absence of pyrexia does not preclude the presence of tubercular toxæmia. When pyrexia does exist, it may be constant, remittent, or intermittent. The inverse temperature which occurs in pulmonary tuberculosis is, in my experience very uncommon.

(Copies of charts showing varieties of temperature met with in the Sanatorium are appended.)



C.J. $[L, S]$ TB + Normal Temperature
Gues. Rati Increased



S. W. $[L_2 S]$ TB + Remittent and Intermittent
Temperature
Pulse Rate: much Increased.

When a pyrexia of the constant or remittent type suddenly comes down below normal in pulmonary tuberculosis, a fatal termination of the case soon results.

Conclusions about Systemic Intoxication in Pulmonary Tuberculosis.

A general toxaemia with the following characteristics/^{occurs} in pulmonary tuberculosis:-

1. It is selective in its action.
2. It bears no fixed ratio to the pathological changes in the lung.
3. It is in inverse proportion to the patient's power of resistance.

Classification.

Some method of classifying cases of pulmonary tuberculosis should be used to emphasize:-

1. The amount of local lesion in the lung.
2. The proportion of systemic intoxication to the local lesion.

With such a classification, it is possible -

- (1) To give a fairly accurate picture of the patient's condition;
- (2) To determine treatment required, as, obviously, cases where the systemic is much greater than the local trouble require rest, and are not fit subjects for tuberculin therapy.

(3) To base a prognosis, or, in other words, to estimate the patient's resistance to the toxæmia.

Guy, in "Tubercle", October 1919, gives a résumé of various classifications, and advocates that some standard should be adopted. I have used Sir R. W. Philip's classification.

L = local lesion.

Turban-Gerhardt scale.

L = Stage I.

L₂ = Stage II.

L₃ = Stage III.

S = Systemic Intoxication.

To indicate proportion, capital and small letters are used.

Thus -

L₁S = Case in Stage I, and the systemic intoxication is not out of proportion to local lesion.

L₁s = Case in Stage I and in fairly good health.

l₁S = Case in Stage I, but shows marked signs of systemic intoxication.

L = healed lesion.

L₁ L₁s L₁S l₁S

Progressively worse case

L₂ L₂s L₂S l₂S

L₃ L₃s L₃S l₃S

Type increasing in Severity.

Now, in the past three years, a greatly increased number of cases has been admitted to the Sanatorium in which the systemic intoxication is vastly out of proportion to what would be expected from the local signs. This is shown by the records for 1917 and 1918, and by my own experience for 1919, when I returned from Active Service abroad.

Another feature is the short duration of illness. A great number of men, who have been four or five years in the Army without a single day's illness, have given a history of feeling in normal health at time of demobilization, but shortly after their return to civilian life, they have become ill, and on examination have been found to be in category 1₂S, 1₃S - in other words, to have galloping consumption. Amongst women, this increase in acute pulmonary tuberculosis does not appear to have taken place.

A Typical Case.

The following is a typical case of the kind to which I refer.

A silversmith, S.W., aet.24. Enlisted M.G.C. 18.8.14. Demobilized 12.5.19. There was no history of any illness previous to or whilst the man was in the Army.

Shortly after demobilization, he began to feel easily tired on exertion, and did not feel inclined to move about much. He saw his doctor, who diagnosed pulmonary tuberculosis, and the man was admitted to Romsley Hill Sanatorium on 7.7.19, in an acutely ill condition. The man looked ill, thin and pallid, and yet the physical signs were not extensive.

Family History.

Father, mother, three brothers and six sisters alive and healthy. No history of tuberculosis in the family.

Symptoms.

Cough - troublesome night and morning.

Sputum - copious, frothy, greenish in colour.

Haemoptysis - nil.

Dyspnoea - marked on exertion.

Weakness

Loss of weight = 1 stone in two months.

Night sweats - present.

Temperature 97° F. - intermittent and remittent.

Pulse - 98 per minute.

General Examination. Patient looked ill, thin and pallid.

Chest. Generally thin and flat, especially flattened left apex, where respiratory movement deficient.

Percussion anteriorly. Note impaired - 2 from apex to 3rd. rib on left side.

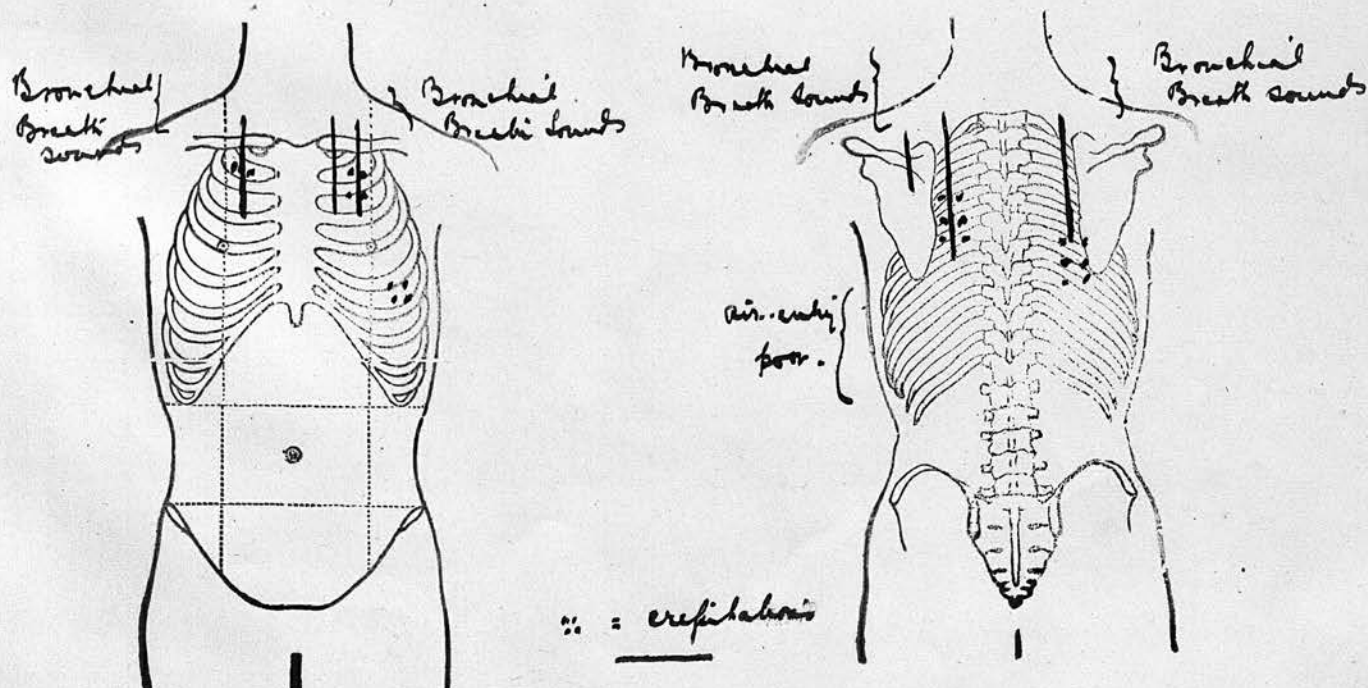
Note - 1 from apex to 3rd. rib right side.

Posteriorly. Note - 2 upper $\frac{1}{2}$ left lung, and - 1 upper $\frac{1}{2}$ right lung.

Auscultation. The breath sounds were bronchial in type in both supra-clavicular spaces. A few crepitations were present in the 1st. space, right side, and on the left side in the 2nd. and 3rd. spaces, also in the lower axillary region.

Posteriorly. Bronchial breath sounds in both supra-scapular spaces, and vocal resonance increased in left supra-scapular space. A few crepitations were present in both inter-scapular spaces.

Air entry was poor in left lower lobe.



From the physical signs, the case was one in Grade II, L₂. The man, however, was seriously ill; in other words, the systemic intoxication was greatly out of proportion to the local signs. His classification was therefore 1, S. Tubercle bacilli were present in the sputum, but not in any great quantity.

Progress of the Case. The man was confined to bed for four months, during which time his temperature was remittent and intermittent up to 102°F. The pulse rate varied from 108 per minute to 146. The physical signs remained practically unchanged, except latterly, when crepitations, probably of a hypostatic nature, were audible at the bases. The cause of death was toxæmia produced by tubercle bacilli.

This type of case is increasing in frequency. It is remarkable how short was the period that elapsed from the time the man was in good health to the time when he was seriously ill. He made no response to Sanatorium treatment.

Effect of War Service on Men previously Consumptive.

It might be urged that such cases were tubercular during service in the Army. I have seen many cases of known tuberculosis, who have served in the Army during the past war, and who, on demobilization, remained well. Prest, in the "Lancet", August 9th., 1919, draws attention

to this fact, that war service did not have a detrimental effect on men with pulmonary tuberculosis.

Explanation of Development of Acute Tuberculosis in Soldiers.

As already stated, numbers of men with no history of illness in the Army are being admitted to the Sanatorium with acute phthisis, soon after their leaving the Army. The explanation appears to be, that these men, having led a healthy and active life for four or five years, have lost their immunity to tuberculosis.

Immunity to Tuberculosis.

Immunity to tuberculosis is known to be of an unstable character. Sahli, in his "Tuberculin Treatment", comments on this fact, and states that no real immunity exists, just as in pneumonia or erysipelas. He gives these facts to support his statement:-

1. The frequent recurrence of tuberculosis, and the persistence of tuberculin sensitiveness after the tuberculosis has been cured.
2. The impossibility of immunizing against tubercle bacilli by chemical toxins.
3. It has been shewn, that even the so-called immunity to tuberculosis, obtained with living

tubercle bacilli by Von Behring, is not a real immunity, but merely an increased power of resistance, due to hyper-sensitiveness.

Morrison Davies, in his book "Surgery of Lung and Pleura", page 144, says:- "The development of Pulmonary Tuberculosis, and the spread of the disease, depends primarily on the degree of resistance of the body to the infection. Some people enjoy complete immunity - in others there is such an absence of resistance that, if exposed to an adequate dose of tubercle bacilli, they succumb rapidly to a fulminating tuberculosis. Immunity against tuberculosis is never very stable, and is affected by changing conditions.

Possible Factors in Increasing Mortality from Pulmonary Tuberculosis.

Influenza.

It is remarkable how few patients attribute the commencement of their pulmonary tuberculosis to influenza - not more than 5% of the cases admitted to the Sanatorium gave influenza as the initial illness, and, even then, it is doubtful whether their so-called influenza was not of a tuberculous nature.

In the Sanatorium, during the first wave of the epidemic, most of the patients escaped the disease

altogether, and in only two cases was there even the possibility of an adverse result from influenza. In the second wave, there were only three definite cases among the patients, one of whom died from the disease. The staff suffered much more severely, and 75% of them contracted influenza in a severe form, one nurse dying of the disease.

The experience here is borne out by reports from other places. In the "Lancet", February 14th., 1920, reports are given from Professor Maurice Fishberg in "The American Review of Tuberculosis", who states: "I have not met a single case of Phthisis during the past year which could be ascribed to have followed Influenza." In the "Paris Medical Journal" for January 3rd., 1920, and the "Zeitschrift für Tuberculose" for December 1919, similar observations are recorded. Influenza, as a factor in the increased mortality from Pulmonary Tuberculosis, can be ignored.

Gas Poisoning.

The possibility of gas poisoning arises in regard to the great majority of ex-soldiers. It is very difficult to get accurate histories from the men, but mostly the gassing has been slight, and it is only the minority of cases who seem to have been evacuated,

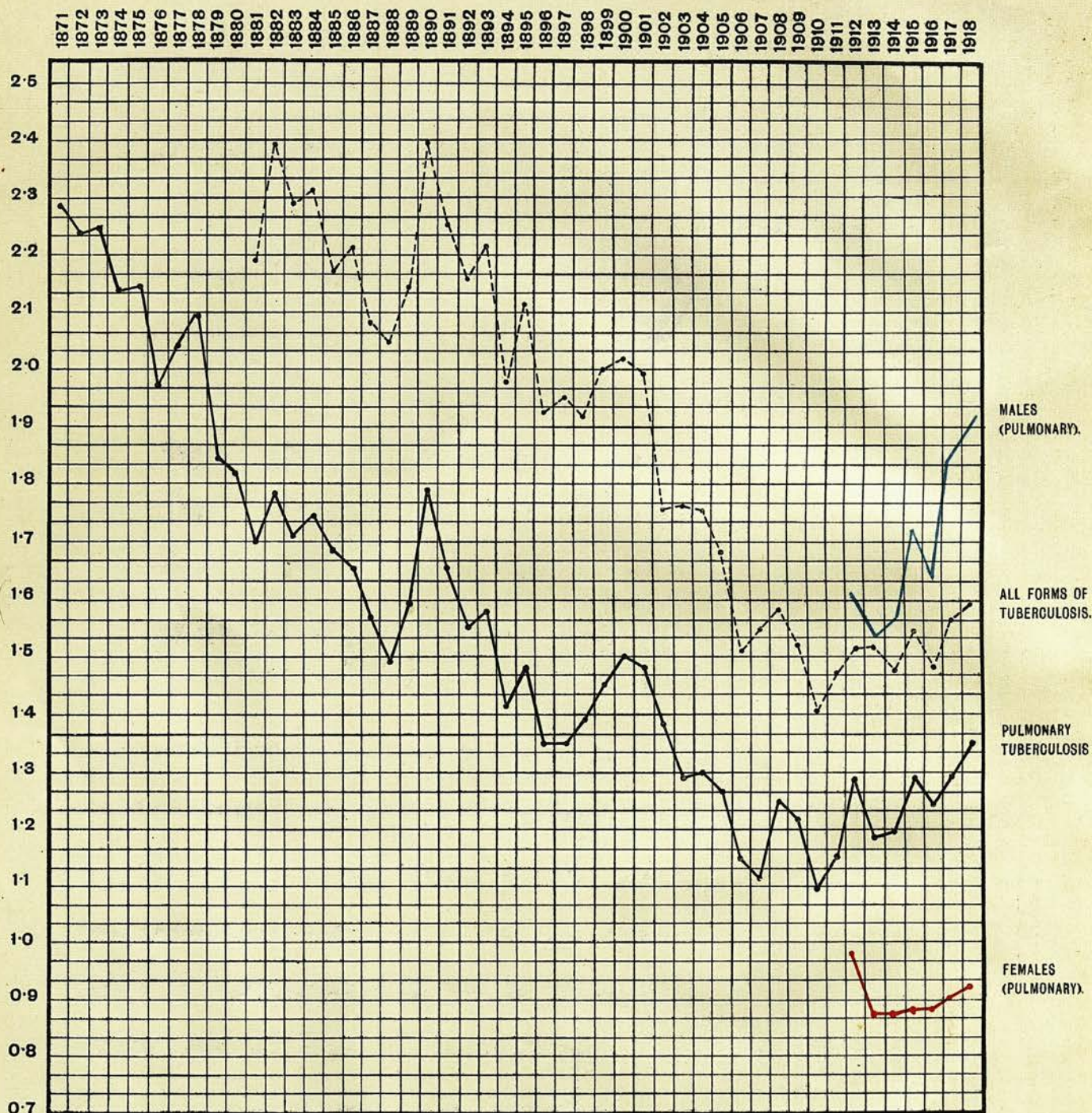
even to a Casualty Clearing Station.

Undoubtedly a certain number of cases must be attributed to gas poisoning, though probably in these it is more a case of the lighting up of latent disease. At a meeting of the War Section of the Royal Society of Medicine, held on February 9th. 1920, while the discussion was on Gas Poisoning, none of the speakers laid any great emphasis on pulmonary tuberculosis as a frequent sequela to gas poisoning. I believe that poison gas was responsible for a certain number of cases of pulmonary tuberculosis, yet do not think it played any large part in increasing the mortality. The sequelae of gas poisoning seem to be more of a bronchitic type, or fibrosis round the lumen of the bronchi.

Lunatics.

Among one class of the population, the death-rate from pulmonary tuberculosis has shewn a great increase, namely, among lunatics. In "The Lancet" of August 9th., 1919, Drs. F. A. Elkins and H. Hyslop-Thompson contribute an article on this subject, and ascribe the increased mortality to the fact that during the war the lunatic asylums were used as Military Hospitals. It is a well-known fact that feeble-minded people are specially liable to

Death Rates in Birmingham from Tuberculosis



ALL FORMS OF TUBERCULOSIS

PULMONARY TUBERCULOSIS

"

"

(MALES)

"

"

(FEMALES)

tuberculosis, and overcrowding would most certainly have a marked effect on the incidence and mortality of the disease.

Comparison of Incidence, as shewn by Notifications, with Mortality.

In the City of Birmingham, the mortality from pulmonary tuberculosis has risen, and yet the notifications have diminished.

	<u>1914</u>	<u>1915</u>	<u>1916</u>	<u>1917</u>	<u>1918</u>
Notifications	3317	3027	3388	3074	2905
Deaths	1059	1141	1107	1169	1171

These figures go to shew that one of two things is happening - either

(1) War conditions have tended to keep up the mortality among those already affected, and indeed increase it (M.O.H's. explanation), or

(2) There is an increase in the number of acute cases. In other words, there is an increasing number of cases where resistance to the tubercular toxin is decreased - that is, where there is decreased immunity.

Relative Mortality among Males and Females.

While the death-rate from consumption has increased, it must be noted in the Birmingham returns that the increase is practically confined to the male population. There has been a slight increase among

females, but among males the increase is most remarkable. (See appended chart.) Now, in a city like Birmingham, there were thousands of women employed in munition factories; and yet, with the war conditions in this country, as regards food and overwork, long hours, and arduous and often unaccustomed occupations, no appreciable effect has been observed on the mortality curve of pulmonary tuberculosis.

From a clinical standpoint, the increase in acute tuberculosis has not occurred among women. Therefore, in seeking a cause for the increased mortality, conditions affecting men especially must be looked to. In Romsley Hill Sanatorium, which does not specially serve for ex-soldiers, over 65% of the men admitted have been in the Army. The possible explanation of the increased mortality is, that return to a primitive life, as in the Army, where men had plenty of exercise and open air, destroyed the immunity to tuberculosis which exists in civil life, and when the men came back to civil life, they were infected by tubercle bacilli, and had little resisting power.

Conclusions.

I. In Pulmonary Tuberculosis, two sets of pathological conditions exist:-

- (a) The local pathological conditions in the lungs,
- (b) The systemic intoxication.

II. The systemic intoxication is not in any fixed ratio to the local lesion.

III. The systemic intoxication is in inverse proportion to the patient's power of resistance, or immunity to the tubercle bacillus.

IV. There has been an increased number of patients with lowered resistance, more especially males, since 1914.

This is shewn by -

- (1) The increased number of cases of acute tuberculosis.
- (2) The non-tolerance of tuberculin.

Prior to 1916, it was the exception, in my experience to meet with cases where absolute non-tolerance to tuberculin was present. In the past two years, it has been very frequent to find men who could not tolerate even small doses of tuberculin as a therapeutic agent; in fact, I found it advisable to temporarily discontinue tuberculin treatment.

VI. As the loss of immunity to tuberculosis is due to actual war conditions, there is every reason to think that the mortality will soon shew a downward tendency again.

* * * * *